

Immediate Effects of Resistance Training on Velopharyngeal Function

Research Thesis

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by

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Abstract

Resistance training is known to be an effective way to strengthen a given muscle. Based on the established principles of the resistance training paradigm, the use of continuous positive airway pressure (CPAP) as a means to strengthen the velopharyngeal closure muscles has shown some promise. During CPAP therapy, speakers exercise velopharyngeal closure muscles by having them work against pressurized air from the CPAP device, which acts as a “weight/resistance.” While there is some evidence supporting the effectiveness of the CPAP therapy for treating hypernasality secondary to cleft palate and traumatic brain injury, information regarding proper treatment dosage is largely lacking. This study examined the immediate effects of individual CPAP therapy sessions with varying pressure levels on the speakers’ velopharyngeal function, with special emphasis on velopharyngeal orifice size and velopharyngeal timing parameters. Results from the study showed that CPAP therapy did not induce any appreciable changes in velopharyngeal orifice size or velopharyngeal timing parameters among healthy individuals. It was also found that varying pressure levels did not alter these speakers’ velopharyngeal valving patterns. No appreciable CPAP therapy effects may partly be attributed to the speakers’ intact velopharyngeal function. That is, velopharyngeal adjustments might not have been necessary for these speakers to maintain proper speech and resonance. It is unclear whether individual CPAP therapy sessions would have similarly limited effects on velopharyngeal function among speakers with velopharyngeal dysfunction, which needs further investigation.

Chapter 1

Introduction

Acoustic aspects of speech production are driven by the underlying aerodynamic processes (Zajac, 2008). Inhalation begins as a result of negative lung pressure generated due to an expansion of the thoracic cavity. Exhalation occurs when passive relaxation forces generate positive lung pressure, which may be further assisted by contraction of expiratory muscles. With regard to speech production, conversion of aerodynamic energy into acoustic energy takes place at multiple levels along the vocal tract. Similar to other articulators, the velopharyngeal valve is responsible for controlling a buildup or release of air pressure for proper sound production. In fact, velopharyngeal valving is an important articulatory gesture, which determines acoustic coupling or decoupling between the oral and the nasal cavities. All oral phonemes in the English language, except for the nasal phonemes (/m/, /n/, and /ŋ/), are produced with oral airflow, by closing off the velopharyngeal port and thus separating the oral cavity from the nasal cavities. Velopharyngeal closure is commonly discussed in relation to three contributors: velar elevation, medial movement of the lateral pharyngeal walls, and anterior movement of the posterior pharyngeal wall. While velopharyngeal closure patterns vary across individuals, the most frequently reported pattern requires significant velar elevation by contraction of the levator veli palatini muscle (Zemlin, 1998). A reverse action is required for the nasal sound production, in which the velopharyngeal port remains open by contraction of velar lowering muscles, such as the palatoglossus and palatopharyngeus muscles (Zemlin, 1998). Different velopharyngeal gestures indeed are the product of synergistic activities of the underlying velopharyngeal muscles (Kuehn & Moller, 2000). This velopharyngeal valving mechanism may not properly function in

individuals born with cleft palate, resulting in excessive nasal resonance (i.e., hypernasality) and/or airflow deviation errors (i.e., nasal air emission).

Intended to reduce hypernasality, Kuehn (1991) proposed a regimented behavioral therapy protocol that utilizes continuous positive airway pressure (CPAP). A CPAP device generates varying levels of continuous positive airway pressure, which has been traditionally used for patients with sleep apnea to keep the airway open while sleeping. Kuehn (1991) suggested the use of CPAP as a therapeutic technique to strengthen velopharyngeal closure muscles. In brief, positive air pressure, delivered to the speaker's nasal cavities during speech, acts a "load or resistance" to the velopharyngeal closure muscles; in other words, positive pressure can be thought of as a weight just as any to strengthen a muscle group which relies on a physical weight. Velopharyngeal closure muscles must work against this pressure load to adequately close off the velopharyngeal port for proper speech and resonance. The "weight" of the CPAP device can be compared to that of any activity intended to increase muscle size, such as weightlifting, if the frequency or amount of activity exceeds those during the normal activation of muscles (Kuehn, 1991). Kuehn also pointed out that the rehabilitation of normal muscles via resistance exercises is an important aspect of muscle rehabilitation, much like techniques used in the field of physical training or physical therapy (1991). In her tutorial review, Clark (2003) provided an in-depth review on CPAP therapy in relation to the principles of resistance training established in the field of exercise physiology. In summary, strength, endurance, and power increases result from hypertrophy of muscle fibers and recruitment of additional motor units, which occur in response to *overload*. The effects of resistance training should be highly specific to the trained behavior, namely *specificity*. Particularly, given that speech is generally a "non-taxing" activity, which is characterized by movements of low to

moderate forces (relative to maximum forces measured during non-speech tasks) and high velocities, those exercises that increase contraction velocity are likely to have the greatest carryover to speech movements (Clark, 2003). In addition, the regimented CPAP therapy also adheres to the principle of progression by increasing the pressure level and session duration as treatment progresses (Clark, 2003; Kuehn, 1991; Kuehn et al., 2002). Thus, the CPAP therapy protocol conforms to the principles of resistance training; if executed properly, CPAP therapy should theoretically induce hypertrophy and increased strength in the velopharyngeal closure muscles (Clark, 2003).

Previous studies have demonstrated the effects of CPAP therapy on velopharyngeal function (Kuehn, 1991; Kuehn et al., 1993; Kuehn et al., 2002; Moon et al., 2007). It is well agreed that the physiologic work required for the normal speech mechanism generally falls into the lower range of effort in reference to the physiologic work required for a non-speech task (e.g., blowing), meaning that speech for normal speaker is a “non-taxing” activity (Barlow & Abbs, 1983; Kuehn & Moon, 1994, Clark, 2003). Kuehn et al. (1993) demonstrated that both speaker groups with and without a history of cleft palate had greater levator veli palatini muscle activities in response to increased intranasal air pressure conditions (i.e., when positive pressure was introduced to the nasal cavities) relative to the zero pressure condition. Specifically, nine subjects, four with and five without cleft palate, were studied in the study using electromyography to see if varying pressure levels from the CPAP device had different effects on the levator veli palatini muscle activity. The levator veli palatini muscle activity was found to be greater for the positive air pressure conditions compared to without for both subject groups, with and without cleft palate. This finding lends empirical support to the use of CPAP therapy as resistance training for the velopharyngeal closure muscles. Based on a series of physiologic

investigations, detailed CPAP therapy protocol with specific pressure and duration prescriptions was suggested (Kuehn et al., 2002), and several studies have reported the effects of CPAP therapy on patients with hypernasality (e.g., Kuehn et al., 2002; Cahill et al., 2005).

The regimented CPAP therapy proposed by Kuehn et al. (2002) consisted of an 8-week CPAP home practice program for 6 days per week with varying pressure levels and session durations: pressure ranging from 4 cmH₂O to 8.5 cmH₂O and session duration ranging from 10 minutes to 24 minutes. Kuehn et al. (2002) reported that 43 speakers with a history of cleft palate and hypernasality had an overall reduction in hypernasality following the 8-week CPAP therapy. These investigators suggested a future study direction: adjusting CPAP therapy pressure/session duration and frequency and closely monitoring patient compliance to increase patient benefits (Kuehn et al., 2002). Similarly, Cahill et al. (2005) used the CPAP therapy to treat hypernasality in three patients with traumatic brain injury with some protocol modifications. The modified protocol consisted of 4 weeks of CPAP therapy for 4 days per week with varying pressure ranging from 4 cmH₂O to 8 cmH₂O and session duration ranging from 10 minutes to 24 minutes. Results of the study illustrated improvement in the patients' resonance characteristics and speech intelligibility, as well as improvement in swallowing function which was unexpected.

Despite some success in CPAP therapy applications for patients with hypernasality (Kuehn, 1991; Kuehn et al., 1993; Kuehn et al., 2002; Moon et al., 2007; Kuehn, Moon, & Folkins, 1993; Cahill et al., 2005), information regarding appropriate dosage, such as pressure levels or session durations/frequency, is largely lacking in the literature. Particularly, the exploration of pressure levels can be compared to a "bodybuilding regimen," especially in relation to intensity of workout. Previous research of resistance training applications in the limb

muscles posed an intensity-related question (e.g., Roman et al., 1993; Trappe et al., 2002); that is, what is the minimum load required to increase muscle mass and overall strength? Likewise, a question may arise regarding the “overload pressure level” required to increase velopharyngeal closure muscle mass and overall strength without muscle fatigue. Thus, this study aimed to examine the immediate effects of individual CPAP therapy sessions with varying pressure levels on velopharyngeal function primarily focusing on velopharyngeal orifice size and timing parameters.

Chapter 2

Methods

Participants

The study was approved by the University Institutional Review Board and informed consents were acquired from the participants prior to their participation. Five adult speakers, three males and two females, without a history of cleft palate or any velopharyngeal dysfunction voluntarily participated in the study (mean age: 21.4, SD: 1.82). None of the participants reported any history of current or recurrent middle ear problems.

CPAP Therapy Protocol

The CPAP device (REMstar Auto A-Flex, Philips-Respironics, Murrysville, PA) is an airflow generator that is connected to a flexible hose and a nasal mask with headgear (ComfortGel Blue Nasal CPAP Mask, Philips-Respironics, Murrysville, PA). All participants underwent a total of 10 CPAP therapy sessions. Each session was 10 minutes long, during which participants were involved in speech activities while receiving randomized pressure air, ranging from 5 cmH₂O to 9 cmH₂O, through the nasal passages. This range of pressure levels was determined to avoid any velar fatigue or exhaust based on previous studies (Kuehn & Moon, 2000; Moon et al., 2007). During each session, pre-recorded speech stimuli were played through the participant's headphones, and the participant was instructed to repeat after each pre-recorded speech stimulus. The same list of speech stimuli suggested by Kuehn et al. (2002), consisting of a series of nonsense words with phonetic makeup of Vowel-Nasal Consonant-Pressure Consonant-Vowel (VNCV) and 12 sentences, was adopted (Figure 1). Detailed information on the rationale of these speech stimuli is described by Kuehn (1991). In summary, the velum is

lowered during the nasal, consonant or second syllable production, but a vigorous velar elevation is required for the upcoming pressure consonant (Kuehn, 1991). Presumably, the levator veli palatini muscle contracts forcefully due to the particular phonetic sequencing of the VNCV words especially against the resistance that is provided from the positive air pressure flow through the nasal cavities (Kuehn, 1991). The levator muscle exerted greater force to overcome the resistance imposed by the heightened air pressure (CPAP). This is an attempt to achieve velopharyngeal closure, a response that eventually could result in increased muscle strength and more complete velopharyngeal closure (Kuehn, 2002). Nonsense words and sentences were repeated until the 10-minute therapy session expired. Participants were allowed to have water breaks if needed.

1. <u>Amtay</u>	26. <u>Amgaw</u>	51. <u>AmpA</u>	76. <u>imfay</u>
2. <u>Ampu</u>	27. <u>untho</u>	52. <u>injaw</u>	77. <u>injee</u>
3. <u>eempay</u>	28. <u>ompu</u>	53. <u>ayngju</u>	78. <u>eengshaw</u>
4. <u>ahngfu</u>	29. <u>ondA</u>	54. <u>eemzee</u>	79. <u>ahmzah</u>
5. <u>ahngzu</u>	30. <u>ahnvee</u>	55. <u>AndA</u>	80. <u>umshah</u>
6. <u>umchah</u>	31. <u>Angchay</u>	56. <u>Engav</u>	81. <u>ongku</u>
7. <u>inzu</u>	32. <u>eengshah</u>	57. <u>eemzA</u>	82. <u>ongkA</u>
8. <u>ingchA</u>	33. <u>ayngju</u>	58. <u>EngkA</u>	83. <u>AmgA</u>
9. <u>Engtho</u>	34. <u>ingjah</u>	59. <u>AmgA</u>	84. <u>ingsho</u>
10. <u>eenjA</u>	35. <u>omshu</u>	60. <u>eenkA</u>	85. <u>Anshah</u>
11. <u>ayngvah</u>	36. <u>Enjah</u>	61. <u>aynggay</u>	86. <u>Engchah</u>
12. <u>omfah</u>	37. <u>unbA</u>	62. <u>ayngjo</u>	87. <u>ayngsah</u>
13. <u>ahnpah</u>	38. <u>imdu</u>	63. <u>aymsho</u>	88. <u>inzah</u>
14. <u>onvay</u>	39. <u>ahngay</u>	64. <u>unsho</u>	89. <u>Amzo</u>
15. <u>ompu</u>	40. <u>ongjo</u>	65. <u>ongbu</u>	90. <u>unzA</u>
16. <u>omgee</u>	41. <u>inday</u>	66. <u>Engpay</u>	91. <u>ungto</u>
17. <u>ingfay</u>	42. <u>ahnpay</u>	67. <u>Empay</u>	92. <u>aymtay</u>
18. <u>Envee</u>	43. <u>aymvu</u>	68. <u>eengfu</u>	93. <u>umdo</u>
19. <u>Empo</u>	44. <u>umthee</u>	69. <u>aymjA</u>	94. <u>Angah</u>
20. <u>aymkA</u>	45. <u>Emshee</u>	70. <u>ayngkay</u>	95. <u>EnchA</u>
21. <u>umbay</u>	46. <u>Envo</u>	71. <u>eengthah</u>	96. <u>Engpah</u>
22. <u>imtah</u>	47. <u>eenbA</u>	72. <u>Amdee</u>	97. <u>Emju</u>
23. <u>Engbah</u>	48. <u>infu</u>	73. <u>eenggu</u>	98. <u>unzA</u>
24. <u>aymvu</u>	49. <u>eemdA</u>	74. <u>impee</u>	99. <u>unfay</u>
25. <u>imthA</u>	50. <u>eengvA</u>	75. <u>ahndah</u>	100. <u>imfo</u>
			101. <u>umkee</u>
			102. <u>eengchu</u>
			103. <u>inggu</u>
			104. <u>omgaw</u>
Mary plans to come.		Find your own pencil.	
Bud took the coat.		Look before you leap.	
He found himself alone.		Inhale between sentences.	
Keep everybody out.		That bus will stop.	
Long samples are needed.		Meaning depends on context.	
She took her brother.		This is a phrase.	

Figure 1. Word and sentence list produced during individual CPAP therapy sessions.

Data Collection and Analysis

A pressure-flow technique (PERCI-SAR, Microtronics, Inc., Chapel Hill, NC) was employed to obtain aerodynamic data from the participants immediately before and after each CPAP therapy session. The time point immediately before a CPAP therapy session will hereafter be referred to as Pre-CPAP and the time point immediately after a CPAP therapy session will hereafter be referred to as Post-CPAP. As described by Warren et al. (1990), the cross-section of constriction (i.e., velopharyngeal orifice) can be estimated based on differential oral-nasal pressure and nasal airflow values. Participants were instructed to repeat the word, “hamper,” in which the velopharyngeal port status is required to rapidly shift from an open to a closed configuration. With the use of “hamper,” there is a well established literature that examined both velopharyngeal orifice size and velopharyngeal timing patterns in individuals with and without cleft palate (Leeper, Tissington, & Munhall, 1998; Warren, Dalston, Trier, & Holder, 1985; Warren, 1964; Warren, Dalston, & Mayo, 1993; Warren, Dalston, Morr, Hairfield, & Smith, 1989; Zajac & Hackett, 2002; Zajac & Mayo, 1996).

Based on speech aerodynamic data acquired at Pre-CPAP and Post-CPAP, velopharyngeal orifice size and two velopharyngeal timing parameters were measured from the middle five repetitions of “hamper.” Figure 2 illustrates the nasal airflow and intraoral pressure changes during the production of “hamper.” Velopharyngeal orifice size was measured during the /p/ segment. Two timing parameters, including 1) the time interval between peak nasal airflow and peak intraoral pressure and 2) the time interval between the onset and the offset of nasal airflow, will hereafter be referred to as Peak Pressure-to-Peak Airflow Duration and Nasal Airflow Duration, respectively. It is presumed that Peak Pressure-to-Peak Airflow Duration best

depicts the timing associated with rapid velar elevation from /m/ to /p/, and Nasal Airflow Duration best depicts the timing associated with velar lowering and elevation for /m/.

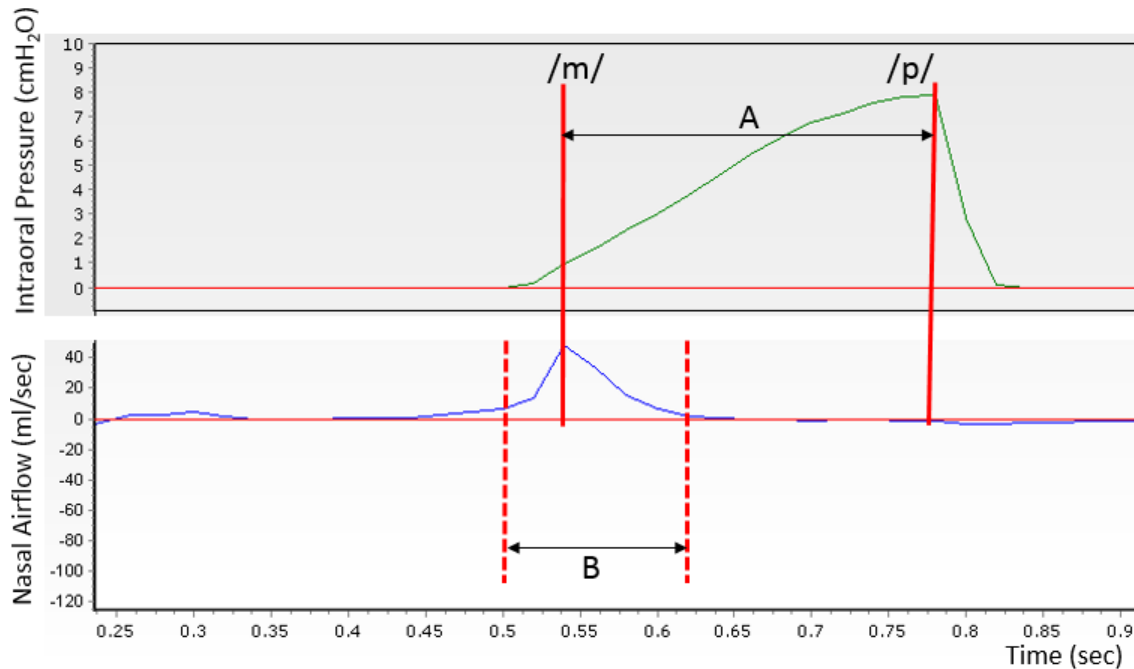


Figure 2. An intraoral pressure and nasal airflow trace for a single production of “hamper.” The time interval between two solid red lines (A) refers to Peak Pressure-to-Peak Airflow duration and the time interval between two dotted red lines (B) refers to Nasal Airflow duration.

Statistical Treatment

An exploratory data analysis was conducted to identify outliers prior to any statistical analysis. Any extreme outliers (26 of 500 samples), falling 1.5 interquartile range below the first quartile or 1.5 interquartile range above the third quartile, were excluded from the subsequent analyses. A series of linear mixed model analyses for the velopharyngeal timing variables were performed using SPSS Statistic 22.0 (IBM Corporation, Armonk, NY). Both Pressure (ranging from 5 cmH₂O to 9 cmH₂O) and Time (Pre-CPAP vs. Post-CPAP) served as within-subjects

fixed factors. The participant parameter was treated as a random factor, and a random intercept was included in each model to account for the clustered observations within each participant. An alpha level of .05 was used for all statistical tests.

Chapter 3

Results

Velopharyngeal Orifice Size

Table 1 provides summary data of velopharyngeal orifice size measured at Pre-CPAP and Post-CPAP in response to varying pressure levels. Although velopharyngeal orifice size estimate of Pre-CPAP was overall greater than that of Post-CPAP, all participants maintained adequate velopharyngeal closure with the velopharyngeal orifice estimate less than .05 cm² (Warren, 1964). The observed velopharyngeal orifice size estimate ranged from 0 cm² to .01 cm².

Table 1. Means and standard deviations (in parentheses) for velopharyngeal orifice size estimates across varying pressure levels.

Pressure (cmH2O)	Pre-CPAP	Post-CPAP
5	.0016 (.001)	.0014 (.001)
6	.0016 (.002)	.0011 (.001)
7	.0011 (.001)	.0018 (.001)
8	.0014 (.002)	.0011 (.001)
9	.0020 (.002)	.0015 (.001)
Mean (SD)	.0015 (.002)	.0014 (.001)

Peak Pressure-to-Peak Airflow Duration and Ratio

Figure 3 provides a series of scatter plots of Peak Pressure-to-Peak Airflow durations (sec) measured immediately before (x-axis) and after (y-axis) CPAP therapy across varying

pressure levels. The diagonal reference line in the scatter plot indicates no change; any data points below the line of no change represent decreased Peak Pressure-to-Peak Airflow duration, and any data points above the line represents increased Peak Pressure-to-Peak Airflow duration. Results from the mixed model analysis showed that Pressure had a statistically significant main effect on Peak Pressure-to-Peak Airflow ($F_{(4,469.022)} = 3.462; p < .05$). Nonetheless, the maximal difference observed between two pressure levels was less than a few hundredths of a second. Neither the Time factor nor the interaction term between Pressure and Time was statistically significant. Given the potential influence of speech rate on the absolute durational measures, a ratio measure of Peak Pressure-to-Peak Airflow duration to word duration was computed for each token (Figure 4). Results showed that, when taking varying speech rates across participants and across different tokens into account, Pressure was found to have no statistically significant effect on the ratio of Peak Pressure-to-Peak Airflow duration. Similarly, no significant main effect for the Time factor or interaction for Pressure by Time was observed. Table 2 provides summary statistics of Peak Pressure-to-Peak Airflow durations and ratios at Pre and Post across different pressure levels.

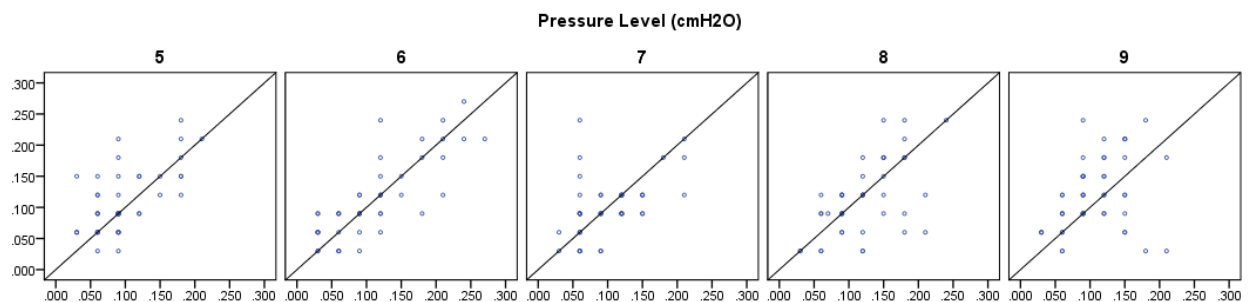


Figure 3. Scatter plots of Pre-CPAP and Post-CPAP for Peak Pressure-to-Peak Airflow Durations (sec) across different pressure levels. The diagonal reference line indicates no change between Pre-CPAP and Post-CPAP.

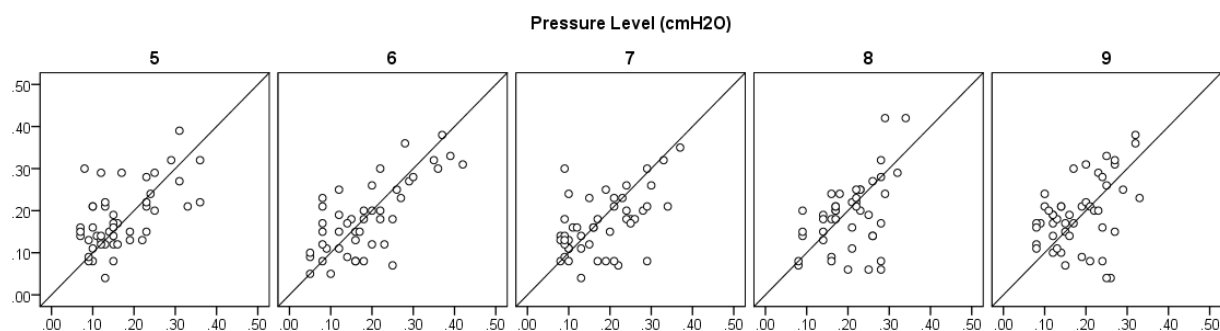


Figure 4. Scatter plots of Pre-CPAP and Post-CPAP illustrating Peak Pressure-to-Peak Airflow ratio (Peak Pressure-to-Peak Airflow Duration / Word Duration) across different pressure levels.

Table 2. Means and standard deviations (in parentheses) for Peak Pressure-to-Peak Airflow durations (Duration) and Peak Pressure-to-Peak Airflow ratios (Ratio) across varying pressure levels.

Pressure (cmH2O)	Variables	Pre-CPAP	Post-CPAP
5	Duration	.097 (.046)	.109 (.050)
	Ratio	.169 (.080)	.182 (.076)
6	Duration	.111 (.066)	.112 (.064)
	Ratio	.186 (.097)	.182 (.087)
7	Duration	.098 (.046)	.099 (.047)
	Ratio	.181 (.082)	.169 (.072)
8	Duration	.120 (.049)	.114 (.059)
	Ratio	.204 (.067)	.190 (.084)
9	Duration	.106 (.044)	.119 (.057)
	Ratio	.184 (.071)	.183 (.080)

Nasal Airflow Duration and Ratio

Figure 5 provides a series of scatter plots of Nasal Airflow durations (sec) measured immediately before (x-axis) and after (y-axis) CPAP therapy across different pressure levels. Results from the mixed model showed that neither Pressure nor Time factors had statistically significant effects on Nasal Airflow Duration. The interaction between Pressure and Time was not statistically significant. In order to account for varying speech rates, Nasal Airflow Duration ratio of absolute nasal airflow duration to word duration for each token was computed (Figure 6). Ratio data further confirmed that Pressure or Time had no statistically significant effects on Nasal Airflow duration and the interaction between the two factors was not statistically significant. Table 3 provides summary statistics of Nasal Airflow durations and ratios at Pre and Post across different pressure levels.

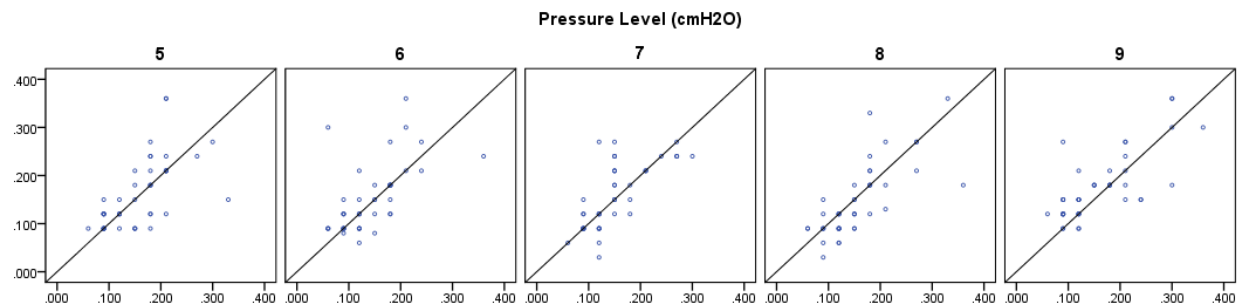


Figure 5. Scatter plots of Pre-CPAP and Post-CPAP for Nasal Airflow Durations (sec) observed across different pressure levels. The diagonal reference line indicates no change between Pre-CPAP and Post-CPAP.

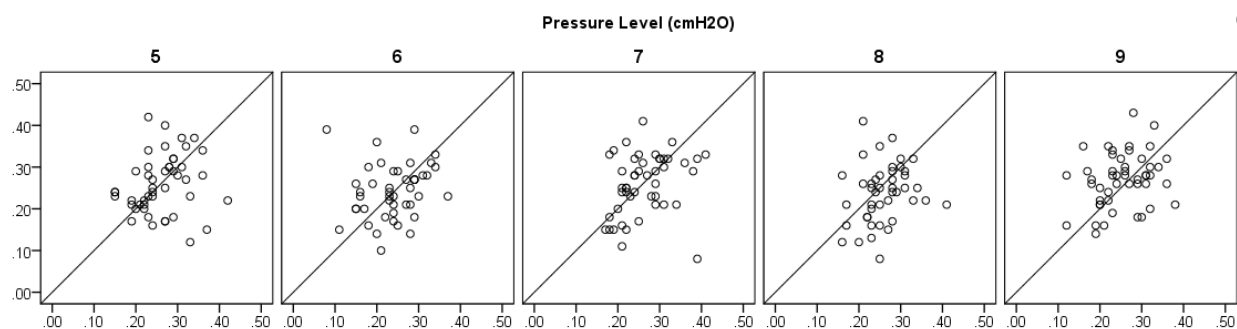


Figure 6. Scatter plots of Pre-CPAP and Post-CPAP illustrating Nasal Airflow ratio (Nasal Airflow Duration / Word Duration) across different pressure levels.

Table 3. Means and standard deviations (in parentheses) for Nasal Airflow durations (Duration) and Nasal Airflow ratios (Ratio) across varying pressure levels.

Pressure (cmH2O)	Variables	Pre-CPAP	Post-CPAP
5	Duration	.156 (.060)	.160 (.070)
	Ratio	.261 (.060)	.259 (.069)
6	Duration	.141 (.059)	.150 (.069)
	Ratio	.239 (.064)	.242 (.065)
7	Duration	.149 (.058)	.157 (.067)
	Ratio	.263 (.062)	.259 (.073)
8	Duration	.155 (.077)	.145 (.074)
	Ratio	.259 (.053)	.238 (.070)
9	Duration	.154 (.073)	.171 (.067)
	Ratio	.256 (.060)	.270 (.064)

Chapter 4

Discussion

The present study examined the immediate effects of individual CPAP therapy sessions with varying pressure levels on velopharyngeal function, primarily focusing on velopharyngeal orifice size and timing parameters. Results from the study showed that individual CPAP therapy sessions did not alter velopharyngeal orifice size or velopharyngeal timing characteristics in healthy speakers, when different speaking rates were taken into account. Furthermore, varying pressure levels delivered through the CPAP device did not induce any differential changes in velopharyngeal orifice size or velopharyngeal timing characteristics. This finding can be related to previous studies (Robbins et al., 2005; Kraaijenga et al. 2015), in which lingual and suprahyoid muscle group exercise programs had no significant effects on swallowing timing parameters in elderly individuals without swallowing problems. Despite insignificant changes in the timing variables, it should be noted that healthy elderly participants benefited from the aforementioned exercise programs by demonstrating increases in lingual and suprahyoid muscle volume, swallowing pressure, and muscular strength measures. It is perhaps that normal swallows by the healthy individuals with intact swallowing abilities could be carried without recruiting additional strength gained by resistance training programs. Similarly, any CPAP therapy-induced gains may likely affect the speaker's capacity in generating maximum possible force for velopharyngeal closure in response to taxing tasks; given that velopharyngeal closure for speech is a non-taxing task, healthy speakers would not need to access this reserve capacity (Kuehn & Moon, 2000; Moon, Kuehn, Chan, & Zhao, 2007). In other words, as healthy participants with intact velopharyngeal function do not need any adjustments to maintain proper speech and resonance, their preferred velopharyngeal timing patterns appeared to be preserved

regardless of resistance training. Future studies need to address to what extent individual's CPAP therapy sessions may alter the velopharyngeal functional capacity in individuals with velopharyngeal dysfunction are necessary.

This study demonstrated an initial attempt to identify appropriate dosage of CPAP therapy, with special emphasis on the pressure levels. With such limited research done on the appropriate dosage of pressure levels and session durations, there is still room for future investigations. Perhaps, the pressure levels utilized in the present study (5 cmH₂O through 9 cmH₂O) with the 10-minutes session duration might have not been “overloading” to induce any appreciable changes in the participants’ velopharyngeal gestures. The immediate effects of individual CPAP therapy sessions may further be probed with increased intensity (high pressure and lengthy session duration), but should be carefully considered as to not fatigue the velopharyngeal muscles. Caution should be exercised when interpreting the findings of the present study in relation to individuals with velopharyngeal dysfunction. Given the intrinsic structural and functional differences between cleft and non-cleft conditions (e.g., muscle volume, thickening/scarring after surgery, muscle fiber type compositions, muscle strength, fatigability, reserve capacity, and tolerance level etc.), the immediate effects of CPAP therapy may have differential effects on velopharyngeal orifice and timing controls in individuals with velopharyngeal dysfunction (Moon et al., 1998; Lindman et al., 2001; Hanes et al., 2008; Moon et al., 2007). Future research with a large sample size, including both individuals with and without velopharyngeal dysfunction, is warranted to determine the immediate effects of CPAP therapy.

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